LETTERS

# Recurrent limp in a young boy

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A child aged 2 years 8 months was referred to our rheumatology unit for evaluation of recurrent acute pain and limited motion of the left hip.

In March 2003 he had been seen at the emergency department of our hospital for a painful, flexed, and abducted left hip. On that occasion, the erythrocyte sedimentation rate (ESR) was 35 mm/1st h, white blood cells (WBC) $8.3\times10^9$/l, and C reactive protein (CRP) 32.7 mg/l (normal value <5 mg/l). Ultrasound of the joint showed mild effusion with normal synovial thickness on the left hip, and a transient hip synovitis (THS) was diagnosed. The pain and limp responded to a 3 day course of anti-inflammatory drug treatment.

One month later, the boy returned again with a limp and pain in the left hip. Because the symptoms resolved over few days with analgesic administration, laboratory tests or ultrasound were not performed.

The child was well up to November 2003, when the hip pain and limping returned without any other musculoskeletal or constitutional symptoms. Abnormal laboratory results were as follows: ESR 34 mm/1st h, CRP 6.5 mg/l, WBC $5.9\times10^9$/l (neutrophils 45.3%, lymphocytes 44.3%), haemoglobin 109 g/l, platelet count $127\times10^9$/l, serum lactate dehydrogenase (LDH) 1216 U/l (normal value <560). Bacterial and viral infections were all excluded. Ultrasound of both hips excluded joint effusion, while an *x* ray examination showed an area of reduced bone density in the left femoral neck. Magnetic resonance imaging with gadolinium showed foci of increased uptake in bone marrow of the left ileum and in periarticular left ischiopubic and ileopubic areas close to the hip. Although a peripheral blood smear did not disclose any abnormality, a bone marrow aspirate analysis showed a myeloblastic leukaemia FAB M2 (fig 1). The boy was then referred to the oncology unit.

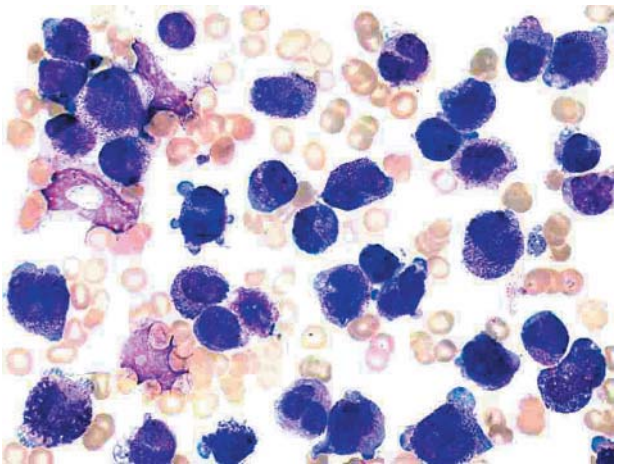

**Figure 1** Bone marrow aspirate showing substitution of normal haematopoiesis with myeloblasts: myeloblastic leukaemia FAB M2.

## DISCUSSION

Limping is a common and challenging problem in children and adolescents. A complete history and thorough physical examination with pertinent laboratory tests and imaging studies are necessary to establish an early diagnosis.[1]

In the absence of constitutional symptoms, THS is the most common cause of sudden onset of limp and flexed and abducted hip in a young boy. The measures of inflammation are normal or slightly raised, joint ultrasound shows effusion on the hip, and *x* ray findings may be normal or show widening of the joint space. The condition may recur and responds to a short treatment with analgesic.[2]

In our patient, the clinical symptoms suggested a diagnosis of THS at initial presentation. After the third episode of limping, an *x* ray examination was recommended to rule out osteonecrosis of the femoral head, a possible complication of THS in about 3% of patients. The increased level of LDH prompted us to investigate further by means of a bone marrow aspirate. In children with musculoskeletal complaints, increased values of LDH may help to distinguish patients with rheumatic diseases from those with malignancies at the beginning of illness when symptoms and other laboratory data are not yet helpful.[3 4]

Our case suggests that malignancy should be considered with a differential diagnosis of recurrent hip pain in children. Recurrent joint symptoms associated with recurrent febrile episodes might also indicate the autoinflammatory diseases spectrum, even though high LDH levels are quite unusual and unexpected in these conditions.

When hip pain recurs, evaluation of LDH could be added to the required blood tests; an increased LDH value should indicate to physicians that they can rule out malignancy.

Acute myeloblastic leukaemia accounts for about 20% of all cases of acute leukaemia in children, resulting in osteoarticular changes in up to 23% of cases. The early diagnosis is a good prognostic feature of this childhood malignancy and misleading and delayed diagnosis can easily occur when classic features of the disease are lacking.[5]

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